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The First International Congress on Forensic Pathology

Brussels, July 1957

We drew attention, just a year ago,[1] to the importance and the need for convening an international meeting of those practising in the field of forensic medicine. The South African Medico-Legal Society was prepared to act as host to such a meeting in Johannesburg and the necessary preliminary investigations were made by that body.

The principle of holding an international gathering found ready acceptance among organizations on whose interest and support the success of such a meeting would depend; but certain practical difficulties arose in connexion with Johannesburg as the venue for the first meeting, largely because an International Congress of Clinical Pathology had already been arranged to take place in Brussels at about the same time as that proposed for the Forensic Congress. Inter-continental efforts are therefore being co-ordinated to make an outstanding success of this first international convention of forensic practitioners.

Those interested in the meeting and desiring further details should communicate either with Dr. Charles P. Larson, Tacoma General Hospital, Tacoma 5, Washington, D.C., U.S.A. or Dr. Francis E. Camps, 37 Welbeck Street, London, W.1., England.

1. Editorial (1956): This Journal, **3**, 1.

Ante-Mortem and Post-Mortem Bruises of the Skin

Their Differentiation

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The question whether a bruise of the skin found at necropsy was caused before or after death arises not infrequently in Courts of Law, when it causes controversy amongst medical witnesses. For this reason the problem has assumed some importance in medico-legal practice. The difficulties will be examined from the point of view of the forensic pathologist, and a few ways in which it may eventually be solved will be suggested.

Evidence of Post-Mortem Bruising

The time of infliction (whether *ante mortem* or *post mortem*) can only be questioned if it is possible for bruises to be produced by blunt force applied to the surface of the body after somatic death. That this can occur must now be readily conceded and the possibility is accepted by the authors of modern textbooks on forensic medicine. For example, Taylor's *Principles and Practice of Medical Jurisprudence*[1] quotes the experience of Christison, who found that blows inflicted on a body not more than 2 hours after somatic death gave rise to appearances on the skin similar to those which resulted from blows inflicted on a person shortly before death. The experiments of Lesser, quoted by Sydney Smith,[2] support these findings in respect of violence applied to the bodies of newly-born infants shortly after death. Glaister,[3] Simpson[4] and Moritz[5] all concede that fluid blood can extravasate from vessels damaged by blunt force applied after death. We have often observed subcutaneous bruises produced in the recently dead (but still warm) body, before rigor mortis has set in.

It is not often that bodies are subjected intentionally to violence after somatic death, but it is easy to envisage ways in which accidental trauma may be sustained.

The movement of a body thrown after assault into rapidly flowing water, the careless transportation of a body on a steel stretcher or even the resusci-

tative efforts of a panic-stricken relative, are some examples of means by which a body could sustain injury. It is important to realize, moreover, that such circumstances are more likely to supervene within the first hour or two after somatic death than much later, and this is precisely the time in which post-mortem bruising is likely to occur in favourable circumstances.

It seems clear, then, that post-mortem bruising of the skin may sometimes occur and, whilst not any blunt force will cause it, the opportunities for its occurrence may arise quite often. The differentiation of ante-mortem from post-mortem bruising becomes, therefore, more than a merely theoretical problem. The sort of case which may cause difficulty is one in which a lesion due to a natural disease process, e.g. coronary artery atheroma, is found which provides a possible explanation for death. If bruises are found on such a body, particularly on the head or neck, suspicion may arise about the circumstances of the death, and an opinion may be required about the time of their infliction.

Criteria for Distinction Between Ante-Mortem and Post-Mortem Bruising

This question does not receive much attention in most medico-legal textbooks in English. Most authors do, however, put forward some suggestions for differentiation.

Extent of Extravasation of Blood

One would not expect the same force to produce as large a bruise after death as it would if administered before death, as the heart is no longer beating in the former instance, and the extent of extravasation would therefore be limited. Many practitioners have witnessed the speed with which a haematoma can form, and it would be surprising if anything like this could occur after death; but whereas the force of the beating heart may result in a much larger bruise in the living body, the post-mortem contraction of blood vessels, which is known to occur, may be sufficient to produce significant extravasation of blood if blood vessels are injured *post mortem*; and, in fact, this is possibly the very mechanism by which post-mortem bruises sometimes become quite prominent. Carscadden[6] observed a rapid movement of blood in the capillaries of a rabbit's lower eyelid removed 10 minutes after the heart had stopped beating. Magnus[7] observed that movement of blood was maintained in the gall bladder and in hydrocele and hernial sacs for from 15—20 minutes after their removal. This movement consisted of an emptying of the arterial side of the vascular system into the more distensible venules. He found that this movement of the blood was not dependent on gravity and that it seemed to be due to an inherent contractility of the arterioles. Furthermore, he found that injury

to a localized area of these tissues caused a local capillary dilatation and redistribution of the blood to produce a localized congestion of the affected part. Whether or not this phenomenon adds to the extent of a post-mortem extravasation of blood, it would still be most surprising if the same force produced the same size of bruise, whether applied before or after death. Theoretically, then, one should be entitled to say that a very large bruise was caused before death. The difficulty is that the converse does not necessarily hold. A smaller bruise is not necessarily a post-mortem one; nor do we know the limits of size of post-mortem bruises. In certain cases, moreover, small bruises may be as significant as large ones.

It may be said that the severe violence which may be necessary to produce even a small post-mortem bruise occurs very rarely. Yet we have seen a bruise almost 1 inch in diameter produced in the scalp of a man (who had died an hour earlier from natural causes) as a result of an impact with the marble top of a post-mortem dissecting table, which occurred when the body was being placed on the table before necropsy.

Finally, one is seldom able to measure the force which has produced a bruise; and even were this possible, we know that the extent of a bruise depends on many other factors besides the force applied, as has been pointed out by Spilsbury.[8]

For all these reasons an assessment of the time (*ante mortem* or *post-mortem*) of infliction of a bruise, which is based on the extent of the extravasation of blood, is liable to be wrong except in the case of really widespread extravasation; and even here unusual post-mortem trauma may have produced the lesion seen at necropsy.

The Infiltration of Tissues by Blood

The supposition here, again, is that it is only under the pressure of the beating heart that blood can be forced in amongst the tissues of the skin and subcutaneous tissues, and that no amount of violence applied after death can reproduce this appearance. On the basis of this assumption it is sometimes said that if the extravasated blood cannot be washed out by a stream of running water played upon the bruise, or if the blood cannot be scraped off with the edge of a sharp knife, then there is evidence that the bruise was caused before death. Dettling, Schönberg and Schwartz[9] have drawn attention to these methods in the distinction between bruises and staining of the tissues by blood escaping from the vessels during necropsy. A stream of water, or scraping, may remove this blood if applied immediately; but it can, apparently, become lodged very rapidly in connective tissue, as Prinsloo and Gordon[10] have shown. The extravasated blood may then resist these methods of removal.

Prinsloo and Gordon have also shown that extravasations of blood may be produced during dissection of the neck by a commonly used technique. This observation is easily confirmed, and it can be seen that the artefacts

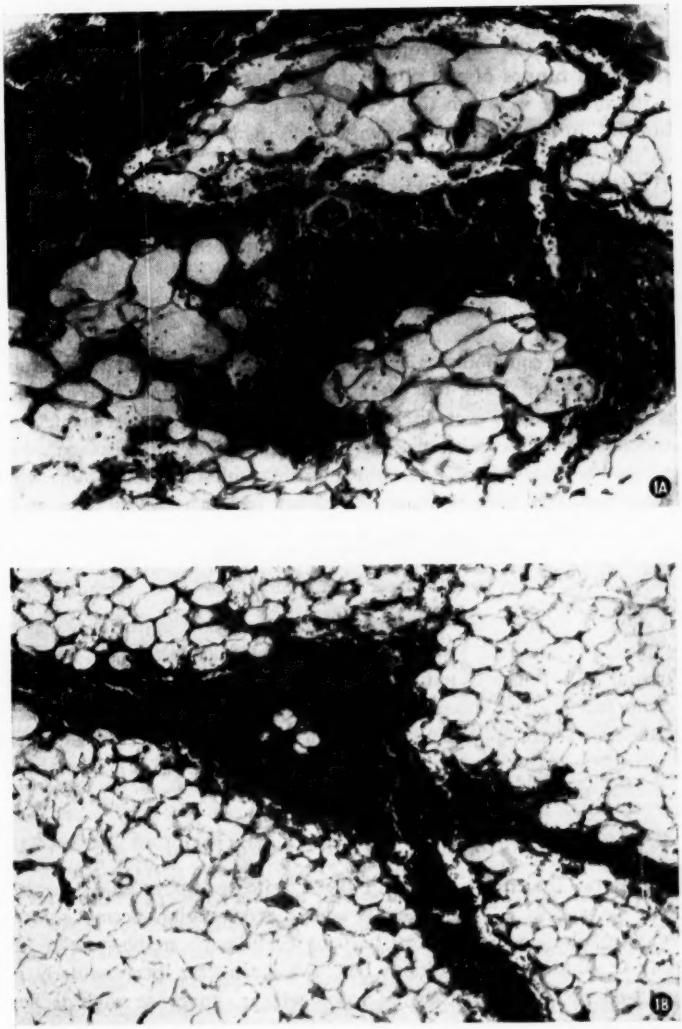

Fig. 1. The bruise shown in Fig. 1a was caused by post-mortem trauma; that in Fig. 1b by trauma sustained 2 hours before death. The way in which the blood lies in the tissues appears to be similar in both bruises.

so produced cannot be distinguished naked-eye from ante-mortem bruising; and that the extravasation of blood resists attempts to dislodge it from the tissues either by scraping or irrigation. Microscopically, also, the appearance of these artefacts is indistinguishable from bruises caused shortly

before death.

Fig. 1 shows the microscopic appearances of known ante-mortem and post-mortem bruises of the skin and subcutaneous tissues. It can be seen that the manner in which the extravasated blood has infiltrated the tissues is similar. In Fig. 1A (post-mortem injury) the blood could not be removed from the tissues by flushing with water.

The foregoing findings suggest that infiltration of the tissues by extravasated blood may occur to such an extent in post-mortem bruises that this factor cannot be used as a reliable guide in differentiating the lesion from one caused before death.

Swelling

Swelling occurs commonly as a result of blunt force applied to the surface of the body before death; it is due to haematoma formation and/or oedema. It is so common, however, for ante-mortem bruises over soft tissues to be unassociated with swelling, that this factor is seldom of assistance. Moreover, in the case of ante-mortem injuries, unless a haematoma forms, swelling may take several hours to develop. In the case of bruising produced shortly before death there may not be time for swelling to develop.

Damage to Epithelium

It is difficult to imagine circumstances in which skin epithelium may sustain visible injury as a result of ante-mortem violence and yet be able to withstand such injury if trauma is applied after death. It is unjustifiable to claim that a given subcutaneous bruise was caused before death because it is associated with epithelial damage. In practice it is not at all uncommon for abrasions to be seen at necropsy which cannot be explained by any mechanism other than post-mortem injury.

Coagulation of Extravasated Blood

If we assume that extravasation of blood can occur only when the blood within the vessels is fluid, then (whether extravasation occurs before or after death) the blood must have been in a fluid state at the time of infliction of the bruise. It is certain that blood can clot intravascularly after death; and there seems to be no reason why it cannot do so if it lies in the tissues. It is true that the centre of a haematoma is commonly fluid, but in the case of the more common bruise, the blood is so intimately associated with the surrounding tissues that we have never been able to tell whether such extravasated blood was in a fluid or a coagulated state. In any case, the state of the blood at necropsy depends upon several factors, as has been pointed out by Mole.[11] The concentration of fibrinolysin in the blood and the post-mortem interval may both affect the findings.

Whether the extravasated blood is clotted or not, can, therefore, have

little significance in this matter, even if it is possible to assess its physical state.

Colour of the Bruise

It is common knowledge that a bruise of the skin undergoes colour changes over a period of days or weeks following its infliction. These colour changes depend on processes which can only occur to any extent during life, i.e. the breakdown of haemoglobin from lysed cells. These changes take several days at least to develop, and so this most helpful feature may be absent when it is most required, viz. to distinguish bruises caused shortly before somatic death from those caused shortly after death. When putrefactive changes are advanced, colour changes in a bruise have, of course, no significance in this problem.

Presence or Absense of Vital Reaction

If life continues after the production of a subcutaneous bruise, the bruise will eventually disappear. The exact mechanism of its disappearance is not certain. Moritz[5] has described the process in some detail and has explained several ways in which extravasated blood cells may be removed from the site of haemorrhage. They may be returned to the blood stream via lymphatic channels; they may be removed after phagocytosis by wandering tissue histiocytes; they may disperse by diffusion into the surrounding tissues; or they may lyse and the products of haemoglobin degradation be removed by lymphatic drainage, phagocytosis or diffusion. Moritz points out that the tissue spaces may constitute an evironment for red cells as favourable as that of the blood stream. Thus intact red cells may be seen at the site of extravasation for several weeks. This may suggest that extravasated blood does not constitute a strong irritant to subcutaneous tissues, and the type of reaction to it may not be the same as that seen in inflammation. Muir and Niven[12] carried out a series of experiments in which they examined the reaction of subcutaneous tissues to fresh homologous red blood corpuscles injected into animals. It is interesting that they found some important differences in rats and mice from the reaction in rabbits. In all cases there was phagocytosis of red corpuscles by local tissue histiocytes, but this occurred to a significantly lesser extent in rabbits. The formation of haematoidin was not observed in the rabbit, in contrast to its formation in the rat and in the mouse. Other noteworthy results were that oedema of the tissues and polymorphonuclear emigration were produced to any extent only when free haemoglobin, not red cells, was the material injected. Lovell *et al.*,[13] investigating the effects of cortisone and ACTH in Man on the dispersal of subcutaneous bruises, were struck by the insignificant part which erythrophagocytosis seemed to play in removing red cells from subcutaneous tissues—an observation confirmed by their studies in guinea pigs. In accordance with the advice in forensic medicine text-

books, we have often examined histological sections of subcutaneous bruises whenever it seemed that the time of their infliction might later become an important question, and we, too, have often been disappointed with the scant information obtainable from the expected cellular response to extravasated blood cells. It is, indeed, mainly this observation which has prompted this paper, and our investigation of this problem, which is still proceeding.

Fig. 2 shows a histological section of the edge of an ante-mortem bruise, which had a yellow-green colour on naked-eye examination and which, we are satisfied, was caused 5 days before death. The absence of a marked inflammatory reaction can be seen; and the illustration shown here did not differ in this respect from other sections taken from different levels in the same bruise.

Fig. 2. The deep edge of a bruise caused 5 days before death. No vascular dilatation or marked polymorphonuclear cell reaction is present. Histiocyte activity and erythrophagocytosis are also not prominent features.

Dettling, Schönberg and Schwarz[9] give details of the vital reaction which occurs to haematomata. They describe vascular dilatation and emigration of leucocytes occurring within the first few hours, if life continues, with the appearance of phagocytes on the third day. In the routine sections of bruises (not haematomata) in our Department in Durban, it has not been possible to confirm the presence of unequivocal vascular dilatation and leucocytic emigration in bruises caused several hours before death. In bruises at least several days old, a few cells showing erythrophagocytosis have usually been demonstrable.

It is submitted, therefore, that the local leucocytic response to free blood cells in the subcutaneous tissues can often be an unreliable guide in the estimation of the time of infliction of a bruise. Apart from any species peculiarity which may exist in Man, there may be other factors which influence the local cellular response considerably. Lovell *et al.*[13] have shown that dispersion of bruises takes longer in persons treated with cortisone or ACTH. They measured the dispersal time as judged by the disappearance of a mauve colour from the bruise. The stress undergone by those whose injuries are examined after death for medico-legal purposes may be an important delaying factor in the reaction of the body to any bruise which may be present. Lovell *et al.*[13] considered that the effects of cortisone and ACTH could not be attributed to impairment of macrophage activity, since even in untreated patients erythrophagocytosis was so inconspicuous. They did observe an increase in the number of histiocytes after the third day, in their experiments on guinea pigs. They did not assess the participation of the lymphatics in bruise dispersion or the part which diffusion of red cells may have played.

Hudack and McMaster[14] investigated the lymphatic participation in cutaneous phenomena in Man. They observed that a dye (injected into the forearm of a normal, resting subject) reached the axillary region within 5—18 minutes after the injection. The lymphatic wall is a semi-permeable membrane; but, whatever the mechanism, it is common to see numerous red cells filling the peripheral sinuses of lymph glands which drain the site of an extensive bruise; and erythrophagocytosis here may be a prominent feature. The question of erythrophagocytosis in lymph nodes is one, however, which bristles with difficulties, and much more information is required before its helpfulness in this problem can be adequately assessed.

The diffusion of red cells, or products of their lysis, into the surrounding subcutaneous tissues may play a vital part in the changes in appearance which occur in a bruise on successive days following its infliction. It is possible that changes in the ground substance of connective tissues may be responsible for the delay in dispersion produced by cortisone. There is no doubt that changes occur in the ground substance as a result of trauma. For instance, pinching of the skin can produce metachromasia of the underlying connective tissue.[15] In any event, the staining procedure would require careful investigation and control before an opinion on the time of infliction of a bruise, based on the presence of metachromasia of the subcutaneous connective tissues, could withstand informed legal cross-examination! It may well be, on the other hand, that the answer to the differentiation of ante-mortem from post-mortem bruises is to be found in changes which occur in the bruised tissues themselves, or in the regional lymph nodes, rather than in the local changes resultant upon extravasation of red cells.

Summary and Conclusions

The criteria commonly applied to distinguish between ante-mortem and post-mortem bruises are described and discussed.

These criteria are often unreliable for the formation of an opinion for medico-legal purposes.

Even microscopic examination of a bruise inflicted up to several hours before death may not make possible its distinction from one caused shortly after death, while the body is still warm.

Red blood cells seem to be readily transported from their point of extravasation to the regional lymph nodes; and subcutaneous connective tissues show staining changes as a result of blunt trauma.

Investigations on these lines may prove more profitable than a study of the local cellular reaction to subcutaneuos bruises.

We wish to thank the Secretary for Health for permission to submit this paper for publication.

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Sex Determination of Human Tissues

From Cell Morphology

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The existence of a sex difference in the nuclei of human tissue cells which are not undergoing active division has been confirmed by a number of workers during recent years and it becomes increasingly apparent that this finding is of clinical value as a diagnostic aid in cases of abnormal sex development. In a previous paper[1] we suggested another important application of chromosomal sex determination which would be the concern of the forensic pathologist, viz. the identification of the sex of small fragments of human tissue. We described how the presence of a distinctive mass of chromatin in the nucleus of female cells can be utilized to determine the sex of such fragments with considerable accuracy for some 2—3 weeks after death, depending on the environment in which the tissue has decomposed. Sex chromatin was found to persist for the longest period after death in tissue which had been immersed in water.

The present account is concerned with the results of a further study of certain aspects of this problem and the consequent additional findings which emphasize the possibilities of its medico-legal applications.

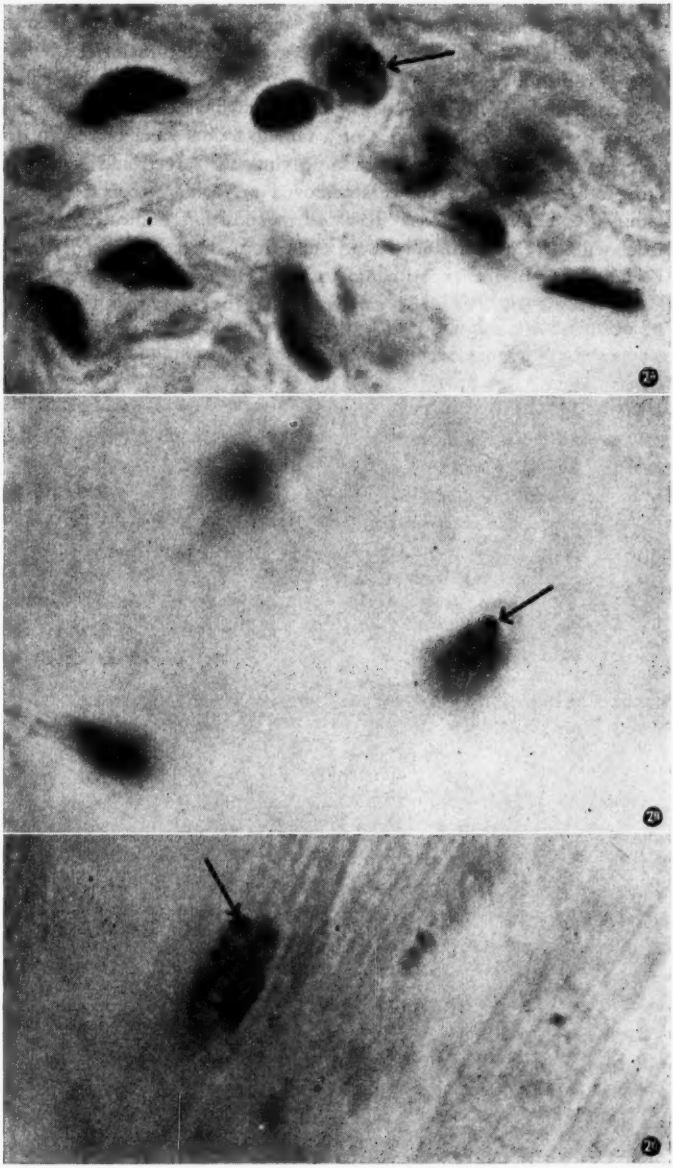
During the course of our earlier investigations it was found that sex chromatin could be seen to best advantage and persisted for the longest period in cartilage cells of fixed tissue, but that this did not appear to be the case with cartilage cells undergoing progressive post-mortem degeneration. Believing, however, that certain tissues may show a greater degree of persistence of nuclear detail than others, we have examined tissues which, by virtue of their situation in the body, may well be affected to a lesser degree by alterations in the environment. As an example, the nuclei of cells in articular cartilage, removed from hitherto undisturbed synovial joints, were stained by routine histological methods and examined. These cells did not reveal any prolongation of the time of sex chromatin persistence when compared with other epithelial or connective tissues which had been similarly treated. As an alternative the cells of the dental pulp were studied, for this tissue is virtually isolated from its surroundings, except by its connections with the dental nerve and vessels through the apical foramina.

A number of teeth from persons of both sexes was obtained immediately after extraction. Some teeth were allowed to remain uncovered in room air; others were immersed in physiological saline, which was renewed at frequent intervals. As it was not possible to obtain teeth *in situ* in the alveolar bone it was considered that storage in saline simulated that condition to some extent. At short intervals the entire pulp of a tooth was removed by crushing the surrounding calcified tissues. The pulp was then

fixed in 10% formol saline, stained with Harris' haematoxylin and eosin, and the cell nuclei examined.

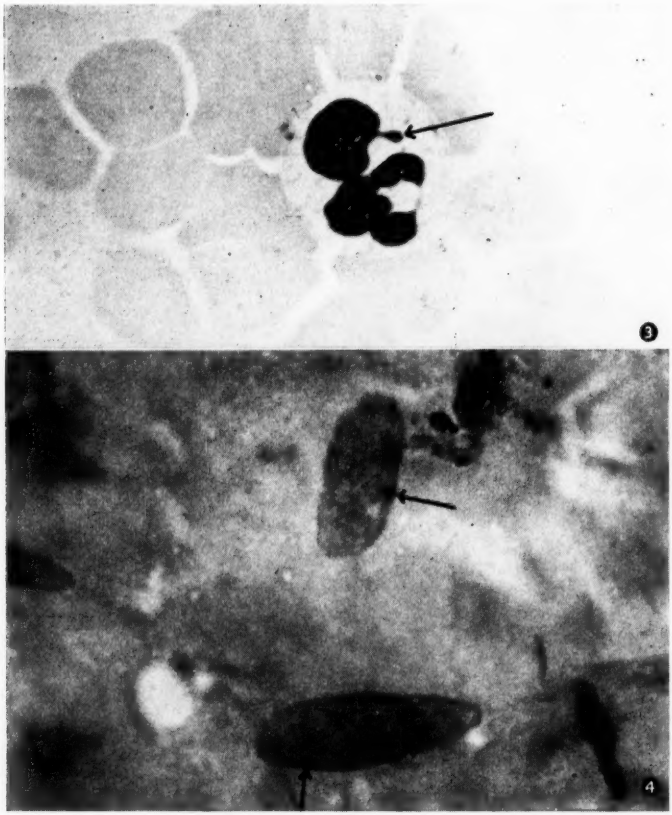
The results were rather unexpected, for in teeth exposed to room air the pulp tissue became rapidly dehydrated and sex determination was impossible only 24 hours after the extraction. In teeth stored in the fluid

medium the period of survival of nuclear detail was also short and did not exceed one week. In the pulp from freshly extracted female teeth



sex chromatin could be seen in many of the numerous spindle shaped fibroblasts. Later these cells became distorted and the nuclei pyknotic so that all chromatin detail was obliterated. It seems, therefore, that in the diagnosis of sex from unfixed tissues, the epithelia, besides being the most readily obtainable, are likely to prove the most suitable material.

As an extension of our previous work on the fate of sex chromatin in various environments, its persistence in tissue which had been buried was considered. Portions of the limbs of a female foetus were buried in direct contact with the soil, at a depth of approximately 2 feet, and small pieces of skin, muscle and cartilage were removed at regular intervals, sectioned and stained as before. Fig. 1A shows, in the positions indicated by arrows, well defined sex chromatin masses in cells from the epidermis of the skin at the beginning of the experiment. In Fig. 1B sex chromatin is similarly indicated in the nucleus of a cartilage cell, the margins of the lacuna



containing the chondrocyte being just visible. After 2 weeks sex chromatin still persisted in all 3 types of tissue and examples are shown in Figs. 2A—2C. The cells in Fig. 2A are from the dermis of the skin. Cells from the dermis are shown, for after the first week the epidermis consistently became detached when the tissue was being cleaned before fixation and staining. Apart from the fact that nuclei of this type are not so numerous in the deeper layers of the skin, their morphology is identical with those normally found in a more superficial position. Fig. 2B shows a well defined sex chromatin mass in a cartilage cell, also after 2 weeks' burial, and in Fig. 2C it is seen in the nucleus of a muscle cell. Note that the cross striations within neighbouring cells are still visible.

We were able to identify sex chromatin with certainty up to 4 weeks after burial, indicating persistence for a longer period than was the case with tissue immersed in water or in atmospheres of varying temperature and humidity. As in the previous paper, we would draw attention to the difficulty of obtaining more than one sex chromatin mass in sharp focus in any particular photograph, due to the high magnifications employed (x 1500).

Attempts were made to determine sex from blood adhering to the scalpel blades which had been used to dismember the foetus. Sex determination from blood is usually carried out by an examination of polymorphonuclear leucocytes, the female cell being characterized by a typical 'drumstick' chromatin body, of which an example is shown in Fig. 3, taken from an adult blood film. It is more difficult to assess the sex of an individual by this method, for the male white blood cell possesses on occasions similar nuclear lobules which are almost identical with the true female 'drumstick'.

In this investigation negative results were obtained from an examination of blood cells, due to haemolysis, the normal picture being absent due to the entire destruction of the white blood cells.

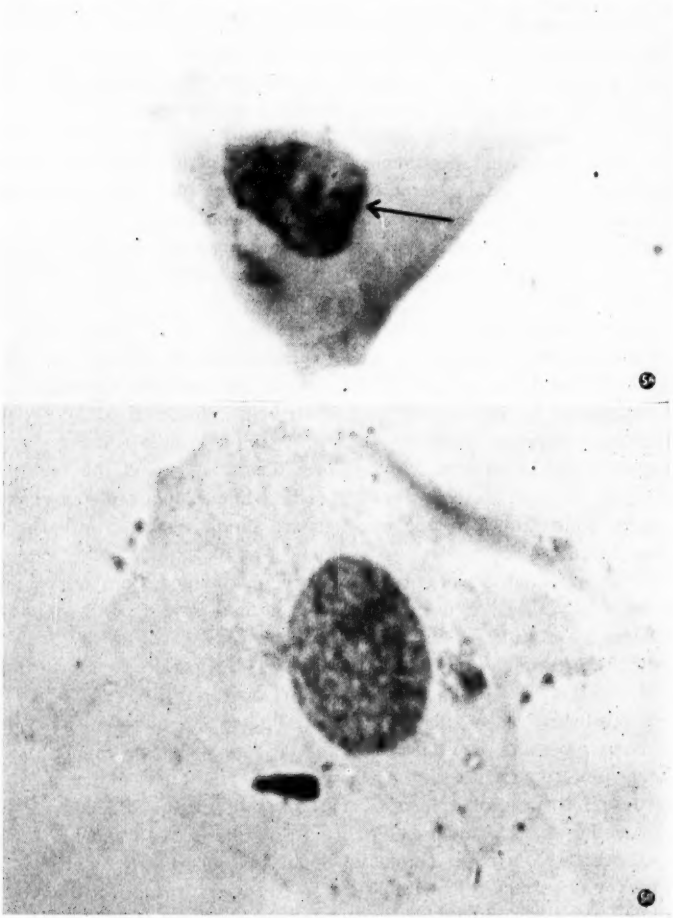
Although examination of the blood was valueless for our purpose, it was found that sex could be determined from epithelial cells which had been transferred to the blade from the skin as the incisions were made into deeper tissues. Examples of such cells are shown in Fig. 4 and the typical plano-convex sex chromatin masses are indicated by arrows. The background consists of a homogeneous mass of disrupted blood cells.

A number of blades used to obtain post-mortem tissue was kept in room air to ascertain if nuclear chromatin detail persists for any length of time in such circumstances. Periodically smears were made of the cellular material and debris on the blades, and stained with cresyl violet. A female cell obtained in this way 3 weeks after the blade was used is shown in Fig. 5A and the sex chromatin mass is indicated at the right hand side of the nucleus. Fig. 5B shows a cell similarly obtained 5 weeks after removal from the tissues. Note that chromatin detail in the nucleus is still

remarkably good and that no sex chromatin is visible. This serves to illustrate two points:

i. If the correct technical procedure has been carried out, sex chromatin in these nuclei is not likely to be an artefact;

ii. While the discovery of one cell possessing sex chromatin is sufficient for the inference that the smear is of female origin, diagnosis of male



smears is more difficult, for a number of cells without sex chromatin would have to be seen to exclude the possibility of female origin, and often only a few epithelial cells can be demonstrated.

We suggest, therefore, that the identification of the sex of material

adhering to instruments or weapons which so often presents considerable, if not insurmountable, difficulties in forensic medicine, may now be a simpler task in view of this additional information concerning the persistence of cell morphology.

A parallel problem is the sexing of human hair, and our examination of a limited number of samples of human hair indicates that here also sex chromatin may be of considerable value. Squash preparations of the hair bulb stained with cresyl violet often show epithelial cells similar to those obtained from the skin. The number of cells obtainable varies with the condition of the scalp and when the hair has been washed recently it is extremely difficult to obtain cells suitable for this method of sex determination.

So far accounts of sex determination have dealt chiefly with histological appearances in normal tissues, but from the medico-legal aspect it seemed important to examine pathological material, in order to discover whether or not previous findings are then applicable. An examination of nuclei in a number of pathological conditions indicates that in many instances the sex of the individual can be readily determined. Although diagnosis is often relatively simple, in tumours showing marked cellular dysplasia (such as the sarcomata) it may be extremely difficult to diagnose sex, because of profound alterations in the nuclear picture, with the appearance of additional masses of chromatin in contact with the nuclear membrane which cannot be distinguished with certainty from sex chromatin.

Conclusions

The results of these additional studies on the persistence of sex chromatin in unfixed tissues show that the sex of small fragments of mutilated tissue, of material adhering to instruments and of human hair, can be determined, under a variety of conditions, from the appearance of nuclear chromatin.

Our findings emphasize that this factual morphological feature of cell nuclei is of importance not only anatomically and clinically, but also for its value as an additional method of sex determination in forensic medicine.

We wish to record our thanks to Prof. G. A. G. Mitchell for his continued support and advice in this investigation.

We would also thank Mr. P. Howarth, A.R.P.S., who was responsible for the photomicrography.

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Adipocere — A Review

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Adipocere is one of the products of decomposition, and consists essentially of a mixture of fatty acids formed by the post-mortem hydrolysis of body fats together with the mummified remains of muscles, fibrous and nervous tissues, and a little soap. Adipocere formation is normally regarded as a phenomenon of late decomposition, because the readily recognizable and fully formed substance may take many weeks, months or years to materialize. This belief is not well founded, and adipocere formation normally commences rapidly after death, its progress being accelerated or retarded by the circumstances and environment of the body.

Adipocere is reputed to have been first mentioned by Lord Bacon[1] but the first authentic description of this substance was undoubtedly that of Fourcroy.[2] Fourcroy's examination and researches took place during the latter half of the 18th century, when a large number of bodies was disinterred from the Cimitiére des Innocens in Paris. His investigations were the subject of a memoir of the Royal Academy of Sciences in 1789. This account is interesting because it contains some references to the current superstitions regarding decomposing bodies, but the most important part of the writing deals with Fourcroy's own observations on adipocere. These were of a high standard, and his macroscopical description of adipocere is as accurate as that recorded in many modern textbooks of forensic medicine.

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He also considered that cholesterol gall stones and animal fat (spermataceti) were the same. Later the substance was re-examined by Chevreul[3] who readily distinguished gall stones from animal fats. He in fact separated in pure state a substance which he called cholesterine which had different properties from fat.

Knowledge concerning adipocere accumulated slowly after the original work of these two French chemists. Fourcroy's work undoubtedly provided the stimulus for further investigations and accounts of these were recorded from time to time. Gibbes[4] found that he could produce the substance in a month in running water. Orfila and Lesueur[5] found that 3—4 months were required for its formation in water during the colder part of the year. An isolated case of adipocere formation was described by Home and Brande.[6] Devergie[7] considered that adipocere formation required a year's immersion in water or 3 years' burial in earth. Casper[8] observed that the substance was formed when a body lay in a damp or wet environment, when colliquative putrefaction is retarded by such an environment, and that the fatter the body, the earlier the colliquative putrefaction ceases.

The current views in the earlier part of the 19th century were briefly that adipocere was an animal soap arising out of the decomposition of body fat (Guy[9]) and its formation occurred more readily in running water.[10, 11] Analyses were made during this period by various chemists whose work was summarized up to about 1860 by Gmelin,[12] and it was found to consist of some 97% of fatty acids, including palmitic and stearic. More recently Rutter[13] examined both human and animal adipocere, and found that human and pig adipocere were similar. He analysed adipocere and found that it consisted essentially of fatty acids, and that the process was complete when the last trace of oleic acid had disappeared.

The chemical composition of pig adipocere, as given by Rutter and Marshall[14] is as follows:

	Per Cent.
Palmitic acid	67·52
Stearic acid	3·3
Oleic acid	5·24
i-Hydroxystearic acid	9·48
o-Hydroxystearic acid	6·32
Stearin and palmitin	1·21
Olein	0·16
Unsaponified matter	0·87
Calcium soaps	4·41
Protein	0·665
Ash	0·578
Humus and undetermine	0·247

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Adipocere — A Review

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Rutter considered that adipocere was a product of the hydrolysis of fats by water under conditions where the time factor and the concentration

of the water are almost indefinitely great, and where the glycerol formed during the hydrolysis is rapidly removed. He considered that bacteria and enzymes played only a secondary role.

Strassman and Fantl[15,16] examined some adipocere which had been many years in water, and found that 80% of the adipocere was ether soluble, and this ether-soluble portion consisted almost entirely of fatty acids of high molecular weights; the remaining 20% consisted of tissue residue and soaps.

Between 1920 and 1940 adipocere received little attention. Sir Sidney Smith[17] made some observations in Egypt, and produced adipocere in the laboratory in new-born infants. He found that adipocere formed as readily in distilled water as in water containing salts. Various authors gave widely differing times for its formation; some have stated that it is recognizable after 3 weeks in the summer, while others maintained that at least 3 months are required. The majority maintained that it required years rather than months before the body became converted to adipocere. Writers of works on forensic medicine generally have slightly differing views on the formation of adipocere, their views arising out of their own personal experiences, which are largely limited to cases removed from the water, as exhumations are an uncommon procedure in this country.

The factors generally accepted as being essential for, or accelerating, adipocere formation, were an excess of moisture, warmth, the presence of skin and subcutaneous tissues and, according to Glaister,[18] a relative diminution of air and micro-organisms.

It became clear during the course of numerous exhumations carried out in Europe following the 1939-45 War that many of the older concepts regarding decomposition in general, and adipocere in particular, were no longer wholly tenable.

Factors influencing Decomposition in Earth Burial

The following were the principal factors found to influence gross adipocere formation after earth burial. Observations were carried out during the exhumations of several hundreds of bodies in north-west Europe during the years 1945 to 1948.[19, 20]

1. *The State of the Body before Death.* As adipocere formation is dependent upon the initial presence of fat, the occurrence of gross adipocere is impossible in a state of extreme emaciation.

It was found that the fatter the body, the better state of preservation after burial, and the more rapidly adipocere formation became recognizable to the naked eye. That large amounts of water were not essential was made apparent by the numbers of relatively thin bodies exhumed that showed adipocere formation in the region of the fat depots and mummification of the limbs. It was also quite clear that the water necessary for the conversion of the body fats into fatty acids was not only obtained from the

environment external to the body, but also from the actual body fluids in both muscles and internal organs. In cases where generalized adipocere was present, muscles and internal organs could normally be found in a mummified form.

2. *Time which Elapsed between Death and Burial.* In many cases investigated in this series, exact factual information was often lacking. However, in one case, where a woman who was well covered with subcutaneous fat, was shot through the neck and immediately buried in a mass grave at a depth of 10 feet from the surface, no gross decomposition or adipocere formation had occurred at the time of her exhumation 26 months later. The exhumation was carried out on a hot day in mid-summer, and the body fat melted in the heat of the sun. All the organs were present and identifiable.

Generally speaking, it was found that the longer the period which elapsed between death and burial, the greater the degree of decomposition. This was particularly marked in the summer months, but was also apparent during the colder months of the year if the body had lain about for many days or weeks before discovery. Decomposition was also found to have been materially accelerated where an autopsy had been carried out before burial.

3. *The Effect of a Coffin.* Many of the bodies exhumed were buried without coffins, but a number were examined who had been buried in crudely constructed coffins of large volume which contained some 3 inches of loose wood shavings in the bottom. These coffins tended to admit water through their lids, and this accumulated in the bottom of the coffin. In these circumstances adipocere formation was usually scanty. The process of colliquative putrefaction continued much longer than in the case of a body buried in the same soil but without a coffin; in these cases adipocere formation was well marked. It should be re-emphasized that these coffins were extremely crude, and besides admitting water, the volume of air contained by the coffin was in many cases twice that of the body.

No bodies which had been buried in good water-tight coffins were exhumed during this series.

4. *The Presence of Clothing or other Covering on the Body.* Adipocere formation was found to be more advanced under clothing, or other coverings over the body, especially if these coverings were close fitting.

5. *Type of Soil.* The part played by various types of soil in altering the rate of decomposition was found to be inconclusive, except in one case where the soil was so well drained that it remained in a state of almost complete desiccation throughout the year. This type of soil was found conducive to mummification.

Occasionally the body was buried in straw or pines at the bottom of the grave and, in cases where this had occurred, the general decomposition

was invariably rapid. Colliquative putrefaction proceeded at a vast rate and, after little more than a year, bodies were reduced to bones and pultaceous fluid, no organs or tissues beyond cartilage being recognizable.

6. *Access of Air to the Body after Burial.* The disturbance of a body in the grave shortly after burial, and before the formation of adipocere or mummification of the tissues, prolonged the period of colliquative putrefaction.

7. *Mass Graves.* Many of the bodies examined were exhumed from mass graves, where a number of bodies varying from 2 or 3 to as many as 50 or 60 had been thrown into a common grave. In these mass graves it was found that adipocere formation was particularly well advanced towards the centre of the bodies, and the rate of decomposition was relatively slow, thus confirming some of Fourcroy's observations.

Before the investigations were undertaken, the knowledge obtained from the literature and from the observations made during the series of exhumations in Europe indicated that the factors summarized below influenced the formation of adipocere.

1. Adipocere formation only occurs where fatty tissue is present. Fatty tissue is naturally essential as adipocere is the result of hydrolysis of the fats. However, it was anticipated before these experiments commenced that, in spite of the views expressed by several writers on the subject, adipocere would form anywhere in the body where fat was present.

2. The earlier adipocere formation commences, the more rapidly will the colliquative putrefaction cease, owing to the fact that adipocere requires water for its formation and, once this formation has begun, will extract water from the internal tissues, thus dehydrating and producing conditions unfavourable to bacterial multiplication.

3. Putrefactive organisms play an important role in adipocere formation. It was long suspected that the hydrolysis and hydrogenation of the body fats after death was not just a simple process depending on the presence of fat and water, and this was apparently confirmed by a unique case which merits a detailed account.

The deceased was a young woman who was shot through the back of the neck into a grave 10 feet deep. An exhumation was carried out 26 months later, and this body showed no gross evidence of adipocere formation. The exhumation was carried out on a hot June day, and the body fats actually melted in the sun. All the internal organs were easily recognizable and showed no putrefactive changes. In this case, where death was instantaneous, and owing to the great depth at which the body was buried, cooling would be rapid and would quickly reach the level at below which organisms would no longer multiply.

4. A large excess of water is not essential to adipocere formation. This does not conform with the views of many writers on the subject, who believe that an excess of water is necessary in order to carry away the

by-products of the hydrolysis of the body fats, such as glycerol.

5. Anaerobic conditions are conducive to adipocere formation.

6. Adipocere formation is retarded by cold, and accelerated by warmth, provided the latter is moist.

Laboratory Investigations into the Formation of Adipocere

The object of these investigations was to subject the existing knowledge concerning adipocere, as summarized above, to a scientific inquiry, and to test theories arising out of this knowledge by controlled experiments.

These investigations fall into two phases:

(i) The actual production of adipocere in the laboratory from human fat, the quantitative analysis of the fatty acid present in the sample of adipocere (the fatty acid being treated as a whole, and the results expressed in the terms of palmitic acid) and the study of various factors which accelerated or retarded its production.

(ii) The examination of naturally occurring adipocere from exhumations, cases of drowning where the body had been in the water for some time, and from other cases where bodies had lain dead for considerable periods before discovery or post-mortem examination.

The first group of experiments were all of a purely qualitative nature, and were undertaken in order to show that adipocere could be produced in the laboratory. These experiments were entirely successful, and the methods adopted for their formation form the basis for the techniques used in the subsequent qualitative investigations. In all the quantitative experiments the material under investigation was examined at regular intervals for fatty acids, and the percentage of fatty acids present was estimated by the method described below, the results being expressed as palmitic acid in view of the original analyses carried out by Marshall and Rutter.[14]

The methods of Marshall and Rutter did not appear entirely satisfactory in some of the later examinations, and it is hoped that at a later date it will be possible, when suitable apparatus is available, to carry out numerous complete quantitative analyses on the adipocere itself, and to ascertain the rate of hydrolysis and hydrogenation of the individual fatty acids rather than of the fatty acids as a whole.

Preparation of Adipocere in the Laboratory. The material used in all the following experiments, unless otherwise stated, was a strip of abdominal fat and subcutaneous tissue removed *post mortem*. Unless the nature of the experiment demanded that the fat should be taken under relatively aseptic conditions, no attempt was made to keep the material clean. In fact, in some of the later experiments the fat was allowed to come into contact with faecal material.

The material was taken from persons who had died sudden natural deaths, e.g. from coronary thrombosis, and was never taken from persons

who were undergoing a course of chemotherapy at the time of death.

A small portion of the material was removed for analysis, and the remaining portion of fat and subcutaneous tissue, which varied between ½—1 inch in width and 4—5 inch in length, was placed in a wide-necked jar which was sealed by a cork. The corks were bored in order that the tissue could be perfused, if necessary, with the bathing fluid, and to allow small quantities of fluid to be withdrawn at regular intervals for pH estimations. The jar was then filled with the fluid to be used throughout the experiment, the pH of the fluid having been taken. The whole container was then immersed in a thermostatically controlled water tank which was maintained at a constant temperature of 37°C. The pH of the fluid was taken at frequent intervals by withdrawing a small quantity of the fluid from the jar and refilling from a reservoir.

At regular intervals the jar was opened and a portion of fat was removed for fatty acid estimation. If the temperature of the water bath was maintained at a constant 37°C., and the other conditions of the experiment, e.g. the type of perfusing fluid, were conducive, it was found that adipocere formed rapidly.

There were, however, a number of technical difficulties which accounted for certain inconsistencies. These were:

1. At a temperature of 37°C. fat is liquified and, as strips of fat and subcutaneous tissue were used, a proportion of the fat escaped into the perfusing fluid and floated to the top of the container. This fat contained quite a proportion of fatty acids, and caused some inconsistencies in the fatty acid estimations throughout the experiment.

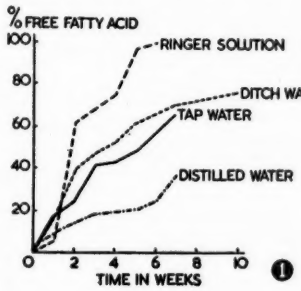
2. It was not technically possible to remove identical portions of material for each analysis. Owing to the length of the original tissue, not all the material was in equal contact with the perfusing fluid and, as fatty acids tended to float away from the tissue irregularly, the portions removed were not always a fair sample.

In the estimations carried out on the material removed, only the ether-soluble portion was examined, the other portion being discarded. The results were calculated from the percentage of fatty acid (expressed as palmitic acid) present in the ether-soluble extracts.

The analysis was carried out as follows:

The portion of material removed was placed in a Sohxlet thimble and then in a vacuum desiccator until there was no further loss of weight. When the material was completely dehydrated, it was placed in a Sohxlet apparatus and extracted for 8 hours with petroleum ether (boiling point from 40—60°C.). The extract was then evaporated to dryness. The flask containing the residue was placed in a vacuum desiccator and dried to a constant weight. The residue was then dissolved in a small volume of neutralized alcohol and titrated with N/10 sodium hydroxide solution, using phenolphthalein as an indicator. From the volume of N/10 sodium hydroxide used to neutralize the solution, the percentage of fatty acids was calculated, and expressed as palmitic acid (1 ml. of N/10 NaOH was equivalent to = 0·0256 g. palmitic acid). In the early stages of each experiment, when only small quantities of fatty acid were anticipated, a micro-burette was used.

1. *The Comparative Effects of Different Solutions on the Artificial Production of Adipocere.* The first experiments were concerned with the study of effects of various waters and solutions on the formation of adipocere. It was not anticipated from the current knowledge on the subject that these would have any marked effect. In these experiments portions of fat complete with skin and subcutaneous tissue were used, and the technique described above was followed.



The solutions used were:

1. Distilled tap water.
2. London tap water.
3. Naturally occurring ditch water.
4. Ringer's solution.

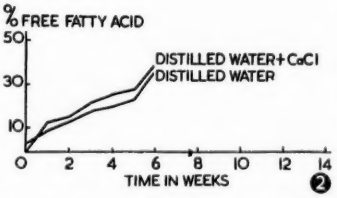
The results of the experiments are shown in Fig. 1.

These experiments were repeated a number of times and on each occasion the results were similar. Each experiment was continued for a period of some 20 weeks, but after the first 2 months the fatty acid estimations became very erratic, owing to the technical difficulties described above.

It will be observed from the results that the hydrolysis of the fat took place most rapidly in the Ringer's solution, and was slow and incomplete in the distilled water. The tap water and ditch water held an intermediate position. It was quite clear from these unexpected results that the presence of electrolytes at least accelerated the formation of adipocere in the laboratory, and that when these electrolytes are in a balanced physiological concentration the hydrolysis was greatly accelerated, becoming complete at the end of some 4—5 weeks.

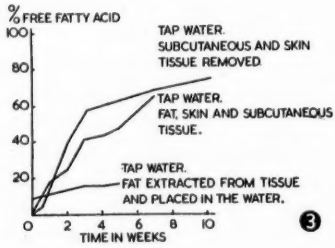
The hydrolysis in distilled water under the conditions of the experiment was slow and in all experiments incomplete. This might appear at first to be opposed to the observations of Sir Sydney Smith, who obtained adipocere by immersing entire foetuses in distilled water. In Sir Sydney Smith's experiments, however, the whole foetus was immersed, and it is reasonable to assume that adipocere formation was accelerated by the diffusion of soluble electrolytes from the foetus into the distilled water, producing an electrolyte concentration sufficient to assist hydrolysis to the stage where recognizable adipocere was produced. In the natural state adipocere is formed in a largely physiological environment, the electrolytes being present in the tissues in amounts varying with the medium in which the body is lying. The experiment suggests that adipocere formation might occur more slowly in water, and also in a large volume of water, than in merely a damp environment at the same temperature, owing to the washing away of electrolytes.

2. *The Comparison of Distilled Water and a Physiological Solution of Calcium Chloride on Adipocere Formation.* The results are shown in Fig. 2.



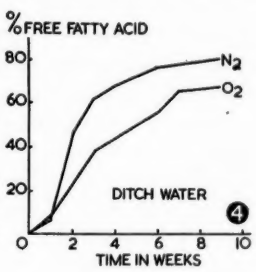
This experiment was carried out in order to confirm, or otherwise, the generally accepted view that the presence of calcium was essential for the formation of adipocere. Experiment shows quite clearly that calcium alone in a physiological concentration has no apparent effect on the formation of adipocere.

3. *The Importance of Skin and Subcutaneous Tissue and the Cell Membranes on Adipocere Formation.* It has long been understood that the presence of skin and subcutaneous tissue is essential for the formation of adipocere. In this experiment a portion of abdominal fat complete with its skin was divided into 3 portions. One portion was left intact; in the second portion the skin and subcutaneous tissue was excised; and the fat from the third portion was extracted with petroleum ether. The 3 portions were then placed in separate jars in tap water, and the experiment conducted as described above. The results are shown in Fig. 3.



It appears indisputable from this experiment that skin and subcutaneous tissues are not necessary to adipocere formation, but it is essential for the fat to be in its cellular state. Fat without skin and subcutaneous tissue formed adipocere as rapidly as complete fat with subcutaneous tissue. However, the extracted fat showed minimal changes in its fatty acid content.

4. *The Effect of Aerobic and Anaerobic Environments on Hydrolysis.* Owing to the lack of agreement on the merits of aerobic and anaerobic environments on the formation of adipocere, a simple experiment was undertaken to test the relative importance of these two factors. A portion of abdominal fat was divided into two and placed in separate jars in the water tank. Nitrogen was bubbled continuously through the ditch water in one jar, and oxygen in the other. The results are shown in Fig. 4.

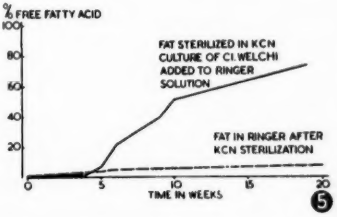


It would appear clear from this experiment that anaerobic conditions are slightly more conducive to adipocere formation than aerobic conditions.

5. *The Importance of Putrefying Organisms on the Production of Adipocere.* In this experiment a strip of abdominal fat and subcutaneous tissue was divided longitudinally, and the two portions were placed in a dilute solution (N/100) of potassium cyanide, and left in this solution for one week in order to kill any bacteria. The fat in this experiment was taken under as aseptic conditions as possible.

After a week the fat was removed from the solution of potassium cyanide and washed continually in sterile distilled water until all traces of cyanide were removed.

The fat was then placed in sterile Ringer's solution in jars. To one jar was added an actively growing culture of *Cl. welchii*, and the experiment conducted from thereon following the standard technique. After a period the fat in the jar to which the *Cl. welchii* had been added showed a steady hydrolysis. The fat in the sterile Ringer's solution, however, showed negligible hydrolysis after 5 months.



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This experiment has been repeated on two further occasions but in none of the subsequent experiments were the results so conclusive as those shown in Fig. 5.

This experiment confirms a theory evolved from the study of bodies exhumed in Europe after the last War, i.e. that some degree of bacterial contamination of the tissues is essential to adipocere formation. The organism was selected because in all cases where bacterial examination had been undertaken on putrefaction, or on naturally occurring comparatively recently formed adipocere, this organism was obtained in almost pure culture.

Observations on the pH. In all experiments undertaken, the pH of the fluid medium fell rapidly to between 4·5 and 5·5. When Ringer's solution was used, the fall was somewhat accelerated, reaching its lowest level in about 48 hours. When tap or distilled water was used, some 7—10 days were usually required for the pH to drop to its lowest level. In solutions to which an active culture of *Cl. welchii* had been added, the fall in pH was such that the organisms were killed by the end of 24 hours, the subsequent hydrolysis of the fat being apparently dependent upon the action of the bacterial enzymes.

The low pH which was found in the laboratory experiments is of interest because whole bodies examined in sealed coffins, where adipocere formation was complete, were strongly ammoniacal, and therefore it would appear that this alkalinity was produced by the decomposition of the body proteins.

Examination of Naturally Occurring Adipocere

Material for these examinations was obtained from 3 principal sources:

1. Routine Coroners' autopsies involving examination of bodies removed from the water, exhumations, and bodies which had been lying dead for varying periods;
2. Material obtained from Professor Thomas, of Ghent University, Belgium, who undertakes a number of exhumations;
3. Bodies removed after the bombing of a crypt of one of the London churches.

The results of the fatty acid estimations of tissues are recorded in Tables 1—4. In all cases the total quantity of fatty acids has been expressed as palmitic acid.

In order to ascertain the amount of fatty acid present in normal post-mortem fat, 5 fresh samples of abdominal fat were examined for fatty acids, and these all contained between 0·5% and 0·59% of fatty acid.

It will be seen at once that, irrespective of the medium in which the body was lying, hydrolysis of the body fat proceeded at a uniform rate, accelerated to some extent by environmental temperature. It will be observed from the table that material had been collected not only from bodies immersed in water or buried, but also from bodies lying in air and in one case, from a mummified child. In some cases parts of internal organs and mesenteric fat were examined, and examination showed conclusively that hydrolysis of the internal fat occurred as rapidly as elsewhere. Owing to the small amount of fat present in the normal liver the hydrolysis was not obvious to the naked eye.

Quite wide variations in the fatty acid content were apparent in portions of fat removed from different parts of the same body. This is because fats of different molecular weights are deposited in different fat depots and, as it was not possible to carry out quantitative analyses of the adipocere, these variations are to be expected from the methods employed.

The material examined fell into 4 groups:

1. Bodies removed from the water;
2. Bodies which had lain in air;
3. Exhumations of recently buried bodies;
4. Exhumations of bodies buried over 100 years.

TABLE 1: Bodies Removed from the Water

	Sex	*Age (Years)*	*How Long Dead*	*Source of Specimen*	*Comments*	*Time of Year*	*% Free Fatty Acid Expressed as Palmitic Acid*
1.	M	51	3/52	Abdomen		Winter	0·9%
2.	F	39	4/52	Abdomen		Spring	6·1%
3.	M	61	6/52	Abdomen		Summer	22·5%
4.	M	21	7/52	Abdomen		Winter	24·5%
5.	M	?	?	(*a*) Thigh			103·3%
				(*b*) Abdomen			72·0%
				(*c*) Liver			120·0%
6.	M	?	?–longer than preceding case	Thigh			115·0%

1. *Bodies Removed from the Water.* The period of immersion varied from 3 weeks to over 1 year. In 2 cases the body was never identified, and the period of immersion was a matter of some conjecture.

It would appear from this limited number of observations that the temperature of the water is of considerable importance in accelerating the formation of adipocere.

In one case the body had lain in cold water in the winter for a period of 3 weeks, and at the end of this time there was very little increase in the free fatty acid content of the tissues. This slow hydrogenation is clearly to some extent due to the rapid initial cooling of the body and the retardation or cessation of bacterial multiplication, especially of those bacteria most concerned with decomposition. In one case (5) where a young man was removed from the river after an unspecified time, adipocere formation was virtually complete. The muscles and the internal organs, however, were still recognizable, and examination of the liver revealed that the liver fat had also become saponified.

Table 2: Bodies which had Lain in Air

	Sex	*Age (Years)*	*How Long Dead*	*Source of Specimen*	*Comments*	*Time of Year*	*% Free Fatty Acid Expressed as Palmitic Acid*
1.	M	18	5/52	Abdomen	Lain in mortuary	Winter	13·5%
2.	M	42	10/52	Abdomen	Fully dressed	Autumn (wet)	72·0%
3.	?	Newborn	8 years	Buttock	Mummified		96·2%

2. *Bodies which had Lain in Air.* Three such bodies were examined. One (2) was a suicide who had hanged himself in a wood in the early autumn. The deceased was fully clothed; it had been a wet autumn and the clothes were saturated. When he was found he was still suspended from a tree and 3 forms of late decomposition had occurred; his arms, which lacked fat in life, were mummified; the thoracic and abdominal organs had putrefied and disappeared. The fat of the abdomen had become hydrogenated, and well recognizable adipocere was present. Analysis revealed 72% free fatty acid expressed as palmitic acid.

The second case (1) was that of an 18-year-old male who was left on a mortuary slab during a cold period of the winter for 5 weeks. He was not refrigerated, and there was no advanced decomposition when the autopsy was carried out. Fat from this case showed 13·5% fatty acid expressed as palmitic acid.

The third case (3) was that of a new-born infant who had been killed by the mother in 1938 and placed in a linen basket, where it became mummified. Autopsy examination took place 2 years later, but the mummified

tissue was not examined for adipocere until 1955, when a portion of buttock was removed. This specimen contained 96·2% fatty acid expressed as palmitic acid, showing that, even in an environment conducive to mummification, hydrogenation of the fats continues at least until desiccation of the tissues is complete.

Table 3: Exhumations of Recently Buried Bodies

	Sex	*Age*	*How Long Dead (Years)*	*Source of Specimen*	*Comments*	*Time of Year*	*% Free Fatty Acid Expressed as Palmitic Acid*
1.	F	24 years	1/12	Abdomen	Coffin	Early spring	25·5%
2.	F	31 years	3/12	(*a*) Breast	Humus soil	Late summer	(*a*) 92·1%
				(*b*) Abdomen			(*b*) 74·0%
				(*c*) Mesentery			(*c*) 73·2%
				(*d*) Thigh (back)			(*d*) 87·0%
				(*e*) Thigh (front)			(*e*) 82·0%
				(*f*) Cheek			(*f*) 99·0%
				(*g*) Liver			(*g*) 96·6%
				(*h*) Buttock			(*h*) 82·3%
3.	F	4 years	9/12	(*a*) Cheek	Buried in clay soil, no coffin	Winter	(*a*) 67·0%
				(*b*) Mesentery			(*b*) 85·0%
4.	?	3 weeks	18/12	Abdomen	Coffin		105·3%
5.	F	18 mths.	21/12	Cheek	Buried in shale	Winter	66·2%
6.	F	14 mths.	4½	Buttock	Coffin (broken)		92·0%
7.	F	20 years	4½	Buttock	Coffin (broken)		97·0%

3. *Exhumations of Recently Buried Bodies.* In this series samples of adipocere from 7 different bodies were examined, the period of burial varying from one month to 4½ years. In one case tissues were examined from 8 different parts of the body.

There were insufficient cases examined to provide much basic information.

In the case of a woman (1) who had been buried for one month, the percentage of fatty acid had reached 25·5%, which is a higher degree of hydrolysis than that found in bodies removed from water after a similar period. However, in this case death was due to an acute infection, and cooling of the body would necessarily have been slower than in those found in water.

In Case 2 (where burial in a shallow grave had occurred some 3 months before) saponification had reached a very advanced stage. The body was buried in late summer, and the months during which it lay in the ground were exceptionally warm.

Cases 6 and 7 were from a mother and child exhumed from the same grave and buried in the same broken coffin. In both the degree of adipocere formation is closely allied.

Table 4: Bodies Buried over 100 Years

	Sex	*Age (Years)*	*How Long Dead (Years)*	*Source of Specimen*	*Comments*	*% Free Fatty Acid Expressed as Palmitic Acid*
1.	F	66	115	Abdomen	Embalmed. Lead-lined coffin intact	127·0%
2.	F	37	116	(*a*) Abdomen (*b*) Liver	Lead-lined coffin intact	(*a*) 84·0% (*b*) 106·0%
3.	F	31	120	(*a*) Mesentery (*b*) Abdomen	Lead-lined coffin intact	(*a*) 93·8% (*b*) 94·3%
4.	F	81	160	Abdomen	Lead-lined coffin intact	92·6%
5.	F	30	167	Abdomen	Lead-lined coffin intact	83·5%

4. *Bodies Buried over* 100 *Years.* These 5 bodies were all buried in a London church which was bombed during the War. The crypt was damaged, and it was necessary to move these bodies. In order to save space the remains were taken from the original coffins and placed in smaller containers. In each case the wooden coffin was hermetically sealed in a lead casing. The lead casing in each case was intact, showing that after the body had been sealed in the lead it was impossible for any air or moisture to reach it from the outside. One of the bodies examined (1) had been embalmed and must be excluded from this series.

Of the other bodies examined, all the abdominal fat in each case showed a percentage of hydrogenation, expressed as palmitic acid, varying from 83·5% to 94·3%, proving that sufficient water is present in a body at death to bring about the hydrogenation of its fats. The adipocere was well recognized as such.

In most cases all the internal organs were readily identifiable, but they were in all cases virtually desiccated. The lungs were reduced to paper thickness, and the heart was similarly desiccated. The anatomical features, however, of both the heart and the lungs were readily identified. Similar changes had taken place below the diaphragm; the livers, however, were by no means so shrunken as the remaining viscera, and in one case examination of the liver showed hydrogenation of the liver fats, and in another case mesenteric fat showed a degree of hydrogenation similar to that of the abdominal fat.

Summary

The foregoing experiments and observations have thrown fresh light on the problem of the post-mortem decomposition of human fatty tissues. Some of the old concepts have been shown to be untenable and others have been modified.

The scope and the detail of the various experiments and analyses were limited by the laboratory facilities and the technical skill available, and it

was not possible to pursue further the several avenues to future research which became apparent as the investigation proceeded. It would appear now that further research in this field must be of a more highly specialized biochemical nature.

It is quite clear that in future full quantitative analyses of the samples of adipocere, both in the laboratory and from decomposing bodies, will be essential.

In these experiments no attempt was made to examine in detail the actual chemical processes which occur, or to differentiate between the relative importance of hydrolysis and hydrogenation, the work being solely concerned with the study of factors influencing the rate of conversion by hydrolysis and hydrogenation of the body fats into fatty acid.

In all the observations recorded, adipocere has been understood to be the product of the hydrolysis and hydrogenation of the body fats. As there are many degrees of hydrolysis and hydrogenation, the presence of adipocere is not necessarily obvious to the naked eye in the early stages of its formation, and its presence could only be ascertained by analysis. The number of investigations was further limited by the time necessary to complete each individual experiment. The same experiments were carried on sometimes for 6 months, in order to ascertain whether any unusual change would occur. It was found after 2—3 months, however, that the fatty acid readings became so erratic (owing to the partial disintegration of the sample in the perfusing fluid, and possibly also to changes in the actual structure of the material) that the latter part of the experiment would appear at present to be of no value.

The information obtained from these observations can be grouped under the following headings:

1. *Origin of Adipocere.* As adipocere consists of the products of the hydrolysis and hydrogenation of fats, the initial presence of fat, and either body or extraneous water, is essential. The formation of adipocere from fat, in its simplest interpretations, is as follows:

By Hydrolysis:

$$\begin{array}{l} C_{17}H_{33}CO-OH_2C \\ C_{17}H_{33}CO-OHC \\ C_{17}H_{33}CO-OH_2C \\ \text{(Olein)} \end{array} + 3H_2O = \begin{array}{l} CH_2OH \\ CHOH \\ CH_2OH \\ \text{(Glycerol)} \end{array} + 3C_{17}H_{33}COOH \quad \text{(Oleic Acid)}$$

By Hydrogenation:

$$\begin{array}{l} C_{17}H_{33}CO-OH_2C \\ C_{17}H_{33}CO-OHC \\ C_{17}H_{33}CO-OH_2C \\ \text{(Olein)} \end{array} + 3H_2 = \begin{array}{l} C_{17}H_{35}CO-OH_2C \\ C_{17}H_{35}CO-OHC \\ C_{17}H_{35}CO-OH_2C \\ \text{(Stearin)} \end{array}$$

$$\underset{\text{(Oleic Acid)}}{C_{17}H_{33}COOH} + H_2 = \underset{\text{(Stearic Acid)}}{C_{17}H_{35}COOH}$$

It was shown conclusively during these investigations that fat anywhere in the body may become hydrolysed and hydrogenated. This was confirmed by every analysis carried out on exhumed bodies. The experiments also showed that at least in the laboratory, the hydrogenation of the fat occurred as readily after removal of skin and subcutaneous tissues, provided the fat remained in its original cellular state. Fat extracted and purified did not hydrogenate under laboratory conditions.

It is not certain whether in certain circumstances fats added to the body after death may or may not hydrolyse to some extent. The examination of the tissues of an embalmed body (oils were used for embalming purposes) suggested from the very high percentage of fatty acid expressed as palmitic acid that a fat of low molecular weight had at least been partly hydrolysed.

Examination of portions of the liver from exhumed bodies showed the presence of fatty acid. This fatty acid was expressed as palmitic acid and, in each case, showed a very high percentage compared with the other tissues examined.

2. *External Conditions Necessary for Adipocere Formation.* It had always been understood that an excess of water was essential for adipocere formation. It was believed that this excess of water was not only necessary for the actual hydrogenation of the fats, but also for the elimination of by-products such as glycerol. Several examinations of bodies in lead-sealed coffins, where the access of water or air from the exterior was impossible, showed conclusively that the average body contains sufficient water in itself for the conversion of the body fats into adipocere. This takes a stage further the suggestion put forward when assessing the changes found during a large series of exhumations.[20] It was believed after these exhumations that although some external water was required for adipocere formation, some of the water used was derived from the muscles and internal viscera, causing mummification and at the same time, by virtue of the dehydration during the hydrogenation of fats, the putrefactive processes were retarded. The laboratory experiments, although not strictly comparable with the natural formation of adipocere, suggest that the rapid fall in the pH may stop bacterial multiplication in the fatty tissues of the body. The manner in which the hydrolysis may occur by dehydration of the tissues was well illustrated in the case of a mummified child whose body fat showed a high degree of hydrogenation.

Although the material examined was limited, the observations made suggest that a damp environment is more conducive to the rapid formation of adipocere than complete immersion of the body in water or, on the other hand, a dry environment. This was supported by the observations made in Europe after the War where, in bodies buried in clothing, the adipocere formation appeared to be more rapid and complete. This is also suggested by Cases 1 and 2 in Table 2, where the rate of hydrogenation was more rapid than in bodies removed from water where they had been

immersed for similar periods at the same time of year. In Case 2, however, the environmental temperature was certainly periodically higher than that of a body in water at the same time of the year. No bodies immersed in sea water were available for examination.

3. *The Mechanism of the Formation of Adipocere.* The complete mechanism of the formation of adipocere from fat, and the various stages through which the fat must pass before complete hydrogenation, may well be shown by future work to be a complicated process involving several intermediate stages, possibly even with the actual splitting of the original fat and fatty acids of similar molecular weights, and it may be found that hydrogenation proceeds more rapidly in certain fat depots because of the local availability of hydrogen donors and the presence of fat-splitting enzymes. The actual size of the individual fat cell may also have considerable bearing on the rate of adipocere formation in that area. In the experimental work undertaken, no attempt was made to pursue these suggestions, and it was only proposed to deal with the hydrogenation in the broadest sense.

It appears likely from the experimental evidence, and from an observation recorded in *Modern Trends of Forensic Medicine*,[21] that bacteria in the early stages of decomposition are essential to adipocere formation. Only a limited number of bacteriological examinations was made on decomposing material where adipocere was present, but in these examinations *Cl. welchii* was invariably the predominant organism. This organism would appear to reach the tissues after death by direct invasion from the bowel. As *Cl. welchii* produces a powerful lecithinase, it is believed that this organism is largely responsible for the hydrolysing and hydrogenation processes, as the fat-splitting enzymes of the skin almost certainly do not survive for more than 2—3 days *post mortem* at room temperature, and it is known that certain bacterial fat-splitting enzymes will function at very low temperatures. As the multiplication of *Cl. welchii* becomes negligible at temperatures below about 70°F., the very rapid initial cooling of the body may prevent the usual adipocere formation. This is suggested by the case quoted above[21] where a woman was shot into a 10-foot grave, covered with a sack and buried immediately. The environment in the grave was moist, as even in summer the water level was only some 12—15 feet below the surface. In this case the preservation was excellent, and there was no visible adipocere formation after 26 months, the body fat melting in the heat of the sun. No facilities were available at the time for making chemical analyses in order to determine the degree of hydrogenation.

Further support is given to this theory by experimental work in which fat sterilized in N/100 KCN remained as fat, whereas a portion of similarly treated fat, to which a culture of *Cl. welchii* had been added, became hydrogenated.

These two observations at least strongly suggest that bacterial enzymes must play an important part in adipocere formation. Furthermore, during

the laboratory experiments it was found that the pH of the fluid medium fell so rapidly that bacterial multiplication ceased within 24 hours, although the hydrogenation proceeds regularly, presumably as the result of the activity of the enzymes.

In the laboratory it was also shown that under identical experimental conditions the presence of electrolytes in physiological concentration accelerated the hydrogenation of fats, and that in distilled water under the same conditions the process was considerably slower. It also appeared that anaerobic conditions were conducive to adipocere formation, although not essential.

Anaerobic conditions produced in the laboratory in relation to an isolated portion of fat are far removed from the natural conditions occurring in a dead body, where (although a body may be exposed to air or water) the majority of the fat is in a relatively anaerobic environment.

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Unnatural Deaths

In a West African Coastal Town (1946-1955)

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The relative frequency of the different forms of violent and unnatural deaths varies considerably from country to country and from district to district within the same country, according to a diversity of factors. This résumé treats of Lagos, the capital city of the Federation of Nigeria, at 6° 30′ N., 3° 25′ E. Lagos is a town having, together with its suburbs, a population of the order of 300,000, including some 5,000 Whites. The town consists of a congested island, surrounded by a lagoon, connected by a bridge with the mainland where densely populated suburbs have developed. The topography of Lagos is shown clearly in Fig. 1.

The figures throughout are derived from the results of autopsies performed on the instructions of the Coroners. The conditions governing the report of deaths to the Coroner under the Coroners Ordinance are, as the Ordinance applies to Lagos, similar to those obtaining in England; also, the Lagos Coroners invariably exercise their right to order a post-mortem examination. This series, covering the 10-year period from 1 January 1946 to 31 December 1955, is probably adequately complete.

It will be seen from Table I that, in all, 1,250 unnatural deaths are under review during this 10-year period, and that the annual totals show a steady rise over the period. The trend is shown by the graph (Fig. 2) in which the figures are plotted on semi-logarithmic paper and the curves smoothed by the shifting mean method. It is clearly the accident rate which has increased rather than the homicide or suicide rates.

Accidents

The distribution of accidents is shown in Table II, and it will be seen that road accidents (504 or 45%) and drowning (341 or 30%) between them account for three quarters of the accidental deaths. The trends of the 3 major groups of accidents (road accidents, drowning and 'other accidents') over the 10 years is shown in Fig. 3. These curves have been smoothed and are plotted semi-logarithmically.

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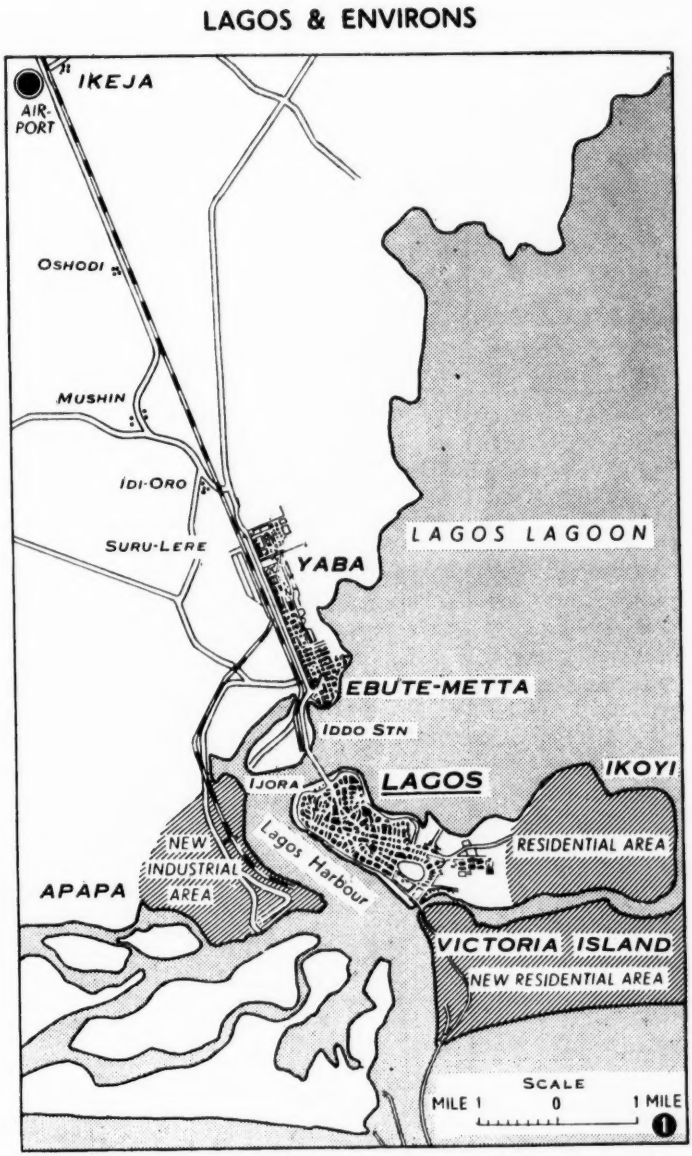

Fig. 1. Lagos and environs. *Note:* Although Victoria Island is marked 'New Residential Area', it has not yet been developed as such.

Table I: Accident: Homicide: Suicide Ratio (1946–1955)

	1946	1947	1948	1949	1950	1951	1952	1953	1954	1955	*Total*
Accidents ..	68 (85%)	84 (88%)	71 (81%)	80 (81%)	104 (93%)	129 (95%)	119 (90%)	133 (92%)	135 (92%)	196 (92%)	1119 (89%)
Homicides ..	11 (14%)	9 (9%)	14 (16%)	13 (13%)	5 (4%)	3 (8%)	8 (6%)	9 (6%)	6 (4%)	7 (3%)	85 (7%)
Suicides ..	1 (1%)	3 (3%)	3 (3%)	6 (6%)	4 (3%)	4 (3%)	5 (4%)	3 (2%)	6 (4%)	11 (5%)	46 (4%)
Total ..	80	96	88	99	113	136	132	145	147	214	1250

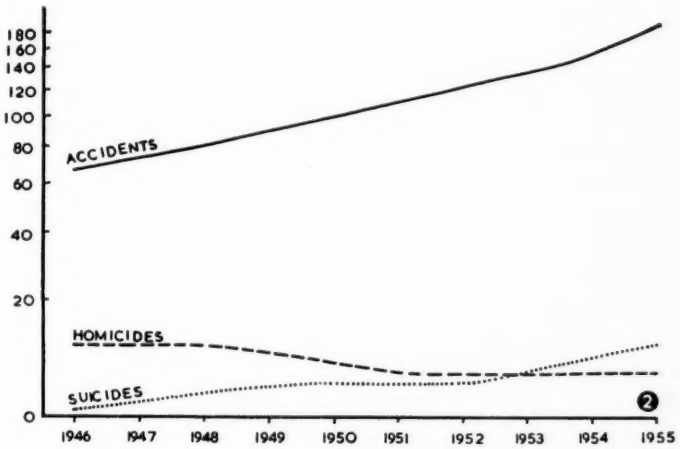

Fig. 2. Semi-logarithmic curves showing the trend of accidental deaths, homicides, and suicides over the years 1946–1955.

The general increase in all accidents is probably due to the increase in population. No figures are available to show accurately the extent of this, but it has been estimated that the population of Lagos has been increasing by about 5% or 6% yearly. The steep incline of the road accident curve during the first part of the 10-year period is due, without doubt, to the rapid increase in motor vehicles on the roads following the relaxation of

Table II: Accidents (1946–1955)

Nature of the Accident	1946	1947	1948	1949	1950	1951	1952	1953	1954	1955	*Total*
Road accidents	19	46	33	31	39	67	43	69	67	90	504
Drowning	21	12	24	17	37	38	40	46	42	64	341
Railway accidents	5	4	1	4	2	4	4	4	5	8	41
Fall from height	6	4	3	7	7	5	8	4	8	6	58
Struck by falling weight ..	3	2	5	6	4	7	3	3	4	18	55
Firearm injury	2	2	1	—	4	1	3	1	—	1	15
Burns and scalds	5	7	2	2	3	1	1	1	2	1	25
Electric shock	5	4	1	5	2	1	8	1	3	4	34
Lightning	—	1	—	4	2	—	1	1	—	—	9
Accidental asphyxia.. ..	1	—	—	—	—	—	—	1	—	—	2
Accidental poisoning ..	1	2	1	4	2	—	1	—	4	3	18
Moving machinery	—	—	—	—	2	1	—	—	—	2	5
Canoe accident (not drowning)	—	—	—	—	—	1	—	—	—	—	1
Sport injuries	—	—	—	—	—	3	2	1	—	—	6
Trampled by crowd ..	—	—	—	—	—	—	5	—	—	—	5
Total	68	84	71	80	104	129	119	133	135	197	1,119

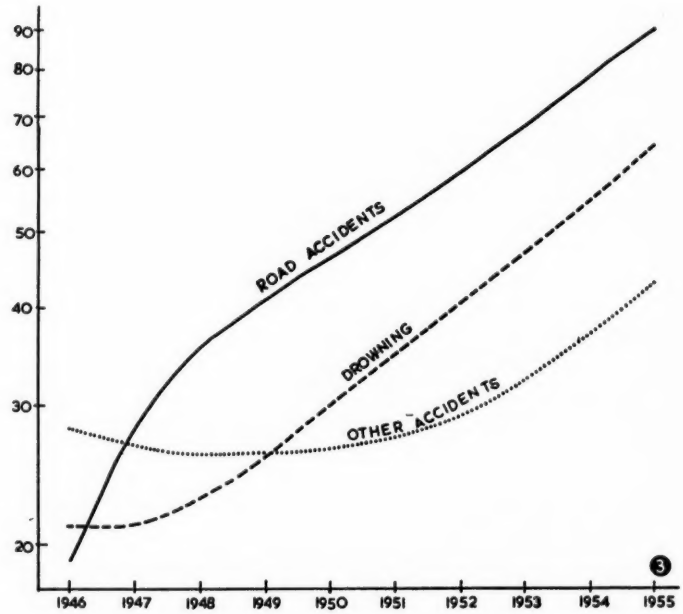

Fig. 3. Semi-logarithmic curves showing the trend of the 3 main groups of accidental deaths, 1946–1955.

war-time restrictions on their purchase. Table III shows the new registrations of motor vehicles each year. While these figures apply to the whole of Nigeria, it is probable that they reflect with reasonable accuracy the trend of conditions in Lagos. The enormous rise in 1947 probably represents the rush to buy motor vehicles when restrictions on their purchase were relaxed. The fall in numbers in the following year and the relatively small increase in 1949 probably indicate that most of the vehicles purchased in the 1947 rush did not have to be replaced.

Table III: New Registrations of Motor Vehicles

Year	*New Registrations*
1946 ..	2467
1947 ..	5439
1948 ..	5114
1949 ..	5309
1950 ..	6240
1951 ..	7198
1952 ..	8368
1953 ..	8592
1954 ..	9895

That road accidents should account for the very high proportion of 45% of all fatal accidents is explained by the narrow, congested streets of Lagos, the generally low standard of driving, and by the lack of road sense shown by the large numbers of pedal cyclists and pedestrians. Negatively, an additional reason may be that Lagos cannot yet be described as an industrial city, and so the incidence of fatal industrial accidents is very low. (Accidents due to moving machinery amount to only 0·4% of all fatal accidents.)

A glance at the map of Lagos shows at once that water enters largely into the life of the community; canoe traffic on the lagoon and in the harbour is heavy, while Victoria Beach is a much-frequented (but notoriously dangerous) bathing resort. It is not surprising, then, that after road accidents, drowning accounts for the next largest group of accidental deaths (30% of the total). There are 2 sources of inaccuracy in the drowning figures: bodies are rarely recovered from the harbour and lagoon until 2 or more days after death and, in the tropical climate of Lagos, they are frequently in an advanced stage of putrefaction, making post-mortem diagnosis of drowning difficult or impossible. In these cases the history has to be relied upon to a great extent, and the pathologist's opinion when expressed to the Coroner has to be very guarded. For the purposes of this survey deaths have been called drowning when the history is clear and when no other cause of death has been found. However, it is probable that any non-drowning deaths which may have been wrongly included in the drowning group are offset by the absence from the Table of

(*a*) Those cases which were in fact drowning but in which such a diagnosis could not be made at autopsy and in which there was no relevant history, and

(*b*) Those genuine drownings in which the body was never recovered. Non-recovery of the body is the rule rather than the exception in drowning deaths occurring at Victoria Beach, where the strong currents and frequency of carnivorous fish (barracuda and sharks) combine to reduce the likelihood of recovery of the bodies.

The 15 accidental fire-arm fatalities are chiefly cases in which a hunter, operating in the bush immediately surrounding the town, has mistaken another human being for an animal. The weapon used in these cases is the Dane gun (a primitive, muzzle-loading cap gun) for which no licence is required. The missiles vary from orthodox lead pellets to small stones, nails and pieces of old iron. Some fire-arm accidents have resulted from these crudely made weapons bursting, thereby killing the operator.

The 18 cases of accidental poisoning are made up as follows:

Acute alcohol poisoning, 6.
Sassy bark, 4.
Cyanide, 3.
Arsenic, 1.
Chenopodium, 1.
Magnesium sulphate, 1.
Carbon monoxide, 1.
Caustic soda, 1.

Sassy bark is the bark of the large forest tree, *Erythrophloeum guineense*, and in the past was used extensively in the conduct of trials by ordeal. Suppression of this custom has been successful, and this use of the bark is very rare to-day in the Lagos area. It is, however, still used in the preparation of native herbal medicines, and such 'pharmacy' leads now and again to death. The bark contains a number of alkaloids (the chief being erythrophloeine) which have an emetic effect but which, after absorption, have a digitalis-like action on the heart.

The cases of accidental cyanide poisoning resulted from the imperfect preparation of cassava for food. The root of this plant (*Manihot utilissima*) contains a cyanogenetic glycoside which has to be removed by thorough scraping and washing.

The cases of chenopodium and magnesium sulphate poisoning were both deaths of children resulting from over-enthusiastic medication.

Coal gas is not used in Nigeria, hence the rarity of carbon monoxide poisoning. The single case in this series was that of a night watchman who sat in a small unventilated hut in company with a charcoal brazier.

The 6 deaths from injuries sustained while engaged in sport involved the following:

Boxing, 2.
Football, 2.
Hockey, 1.
Polo, 1.

Homicides

A considerable number of the road accidents should, strictly speaking, be included under this head as they resulted in convictions for manslaughter. The perpetrators of the 85 deaths recorded here, however, were all initially charged with murder (in those cases in which arrests were effected) although in some cases this charge was reduced by the Court to one of manslaughter.

Table IV shows that a sharp cutting instrument is the weapon of choice,

Table IV: Homicides (1946–1955)

Method Used	1946	1947	1948	1949	1950	1951	1952	1953	1954	1955	*Total*
Blunt instrument	2	3	2	2	—	—	4	3	—	—	16
Sharp instrument	3	1	4	2	1	3	4	2	3	4	27
Stabbing	3	2	2	1	1	—	—	2	2	1	14
Manual strangulation ..	1	1	1	3	—	—	—	—	1	—	7
Strangulation with ligature	1	1	2	5	—	—	—	—	—	—	9
Rape (shock and haemorrhage)	1	—	—	—	1	—	—	—	—	—	2
Smothering	—	1	1	—	—	—	—	—	—	—	2
Firearm injury	—	—	1	—	1	—	—	—	—	—	2
Abortion and unlawful surgery	—	—	1	—	—	—	—	2	—	1	4
Homicidal drowning ..	—	—	—	—	1	—	—	—	—	1	2
Total	11	9	14	13	5	3	8	9	6	7	85

possibly due to the fact that the matchet is a tool to be found in the possession of practically every man, woman and child.

The 4 cases included under the heading *Abortion and Unlawful Surgery* comprise 2 cases of instrumental abortion, the other 2 being deaths directly resulting from surgical procedures performed by unqualified practitioners. One of these operations was uvulectomy and the other was the amputation of supernumerary fingers. In both cases haemorrhage was the cause of death.

Suicides

Of the 46 cases seen, 5 (3 poisoning, 1 hanging, 1 shooting) were non-Africans. The incidence of the different methods used is given in Table V. Hanging is clearly the method preferred.

Table V: Suicides (1946–1955)

Method Used	1946	1947	1948	1949	1950	1951	1952	1953	1954	1955	*Total*
Hanging	1	1	—	3	3	1	3	2	1	6	23
Burns	—	1	—	—	—	—	—	—	—	—	1
Incised wounds	—	1	—	1	—	—	—	1	—	2	5
Firearm injury	—	—	2	1	—	2	—	—	2	—	7
Drowning	—	—	1	1	—	—	2	—	—	—	4
Poison	—	—	—	—	1	1	—	—	3	3	8
Total	1	3	3	6	4	4	5	3	6	11	46

The agents used in the suicidal poisoning cases were:

Cyanide 3.
Lysol, 1.
Caustic soda, 1.
Carbromal, 1.
Nitrous oxide, 1.
Resochin, 1.

I am grateful to Dr. G. T. Barnes of the Pathology Department, Lagos, for allowing me to have access to those post-mortem records kept in his laboratory, to the Director of Federal Surveys for the map (Fig. 1) and to the Chief Medical Adviser to the Federal Government of Nigeria for permission to submit this paper for publication.

Notes and News

Post-Graduate Course in Forensic Medicine

A Post-graduate Course in Forensic Medicine was arranged by Dr. Francis E. Camps, Reader in Forensic Medicine, University of London, at the London Hospital Medical College.

Dr. Camps organized this course to present a proper medico-legal approach to hospital pathologists who were required to perform autopsies on persons dead from non-natural causes and who subsequently had to give evidence about their findings in court.

As many of these hospital pathologists had not received special post-graduate training in forensic medicine, there was considerable need for such instruction and this was attested by the large number of pathologists who attended the course from all parts of the United Kingdom.

The meetings were held on 29 and 30 June 1956.

The following topics were dealt with *inter alia*:

The Medico-Legal Post-Mortem Examination: *External Appearances* (F. E. Camps);

Why Do they Die? (F. E. Camps);

Anoxia (B. G. B. Lucas; Department of Anaesthetics, University College Hospital);

Anaesthetic Deaths: *Deaths Associated with Therapeutic or Operative Procedures* (F. E. Camps);

Time of Death and Post-Mortem Changes (A. C. Hunt);

Identification; Anoxial Deaths; Strangulation; Hanging; Obstruction to the Air Passages (F. E. Camps);

Abortion (R. D. Teare);

Dental Identification (R. Fearnhead);

Wounds: *Their Examination and Interpretation* (F. E. Camps);

Firearm Injuries (J. Friedman);

Common Poisoning (F. E. Camps);

Domestic and Industrial Mishaps (F. E. Camps);

Deaths Associated with Modern Warfare (Lt. Col. Lundie, R.A.M.C.).

During luncheon on the second day the visitors were divided into two groups, Dr. Francis E. Camps and Dr. A. C. Hunt each acting as host to one of the groups. A special subject was discussed at each table. Dr. Camps' group ventilated various aspects of the *Teaching of Forensic Medicine* and Dr. Hunt's table debated *Research in Forensic Medicine*.